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=> s p53 and review/dt

L1 4534 P53 AND REVIEW/DT

=> s l1 and py<1997

L2 1340 L1 AND PY<1997

=> s l2 and p53/ti

L3 273 L2 AND P53/TI

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L4 273 DUPLICATE REMOVE L3 (0 DUPLICATES REMOVED)

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L4 ANSWER 1 OF 273 MEDLINE on STN

AN 96306066 MEDLINE

DN PubMed ID: 8763583

TI [P53 antibodies: a new method for the analysis of alterations of the p53 gene: application to breast cancer].

Les anticorps anti-p53: une nouvelle methode d'analyse des alterations du gene p53: application au cancer du sein.

AU Soussi T; Peyrat J P; Lubin R; Bonnetterre J

SO Pathologie-biologie, (1996 Apr) 44 (4) 232-4. Ref: 13

Journal code: 0265365. ISSN: 0369-8114.

CY France

DT Editorial

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LA French

FS Priority Journals

EM 199610

ED Entered STN: 19961219

Last Updated on STN: 19980206

Entered Medline: 19961028

AB Alterations in the p53 gene are found in 20% to 40% of breast cancers and are generally associated with factors of adverse prognostic significance. In most instances, point mutations modify the confirmation of p53, causing the gene to accumulate in the nuclei of tumor cells. These alterations can be detected via molecular analysis or immunohistochemical methods. More recent studies have demonstrated that accumulation of the p53 protein in tumor cells may induce an immune response with presence of anti-p53 antibodies in the serum of cancer patients. Assaying serum anti-p53 antibody is a new approach to investigation of the status of the p53 gene in a tumor.

L4 ANSWER 2 OF 273 MEDLINE on STN

AN 97000048 MEDLINE

DN PubMed ID: 8843191

TI Strange bedfellows in even stranger places: the role of ATM in meiotic cells, lymphocytes, tumors, and its functional links to p53.

CM Comment on: Genes Dev. 1996 Oct 1;10(19):2401-10. PubMed ID: 8843193

Comment on: Genes Dev. 1996 Oct 1;10(19):2411-22. PubMed ID: 8843194

Comment on: Genes Dev. 1996 Oct 1;10(19):2423-37. PubMed ID: 8843195

AU Hawley R S; Friend S H

CS Department of Genetics, University of California at Davis, 95616, USA.

SO Genes & development, (1996 Oct 1) 10 (19) 2383-8. Ref: 49

Journal code: 8711660. ISSN: 0890-9369.

CY United States

DT Commentary

Journal; Article; (JOURNAL ARTICLE)

General Review; (REVIEW)

(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 199611

ED Entered STN: 19961219

Last Updated on STN: 19961219

Entered Medline: 19961127

L4 ANSWER 3 OF 273 MEDLINE on STN  
 AN 97194830 MEDLINE  
 DN PubMed ID: 9042268  
 TI The dual role of mutant **p53** protein in chemosensitivity of human cancers.  
 AU Mueller H; Eppenberger U  
 CS Department of Gynecology, University Hospital Basel, Switzerland.  
 SO Anticancer research, (1996 Nov-Dec) 16 (6B) 3845-8. Ref: 25  
 Journal code: 8102988. ISSN: 0250-7005.  
 CY Greece  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA English  
 FS Priority Journals  
 EM 199703  
 ED Entered STN: 19970407  
 Last Updated on STN: 19970407  
 Entered Medline: 19970327  
 AB Mutational loss of **p53** tumor suppressor functions has been observed in a wide range of neoplasms and was associated with either enhanced or decreased chemosensitivity of affected tumors. The dual role of wild-type **p53** as a DNA repair initiator and a trigger for apoptosis raises the possibility that appropriately designed chemotherapy could be selectively applied against **p53**-defective tumor cells. The cytotoxic effects of DNA-crosslinking chemotherapeutica such as cisplatin could be enhanced by mutated **p53** which is no longer able to repair drug-induced DNA damage. In contrast, DNA synthesis blockers such as fluorouracil can induce apoptosis through **p53**-dependent mechanisms. Thus, loss of **p53** functions results in decreased sensitivity to this type of drugs. Clinical studies will reveal the role of aberrant **p53** in the efficacy of chemotherapy for individual patients.

L4 ANSWER 4 OF 273 MEDLINE on STN  
 AN 96344733 MEDLINE  
 DN PubMed ID: 8741682  
 TI Role of the **p53** gene in apoptosis.  
 AU Takahashi R; Shinohara H  
 CS Department of Pathology and Tumor Biology, Graduate School of Medicine, Kyoto University.  
 SO Nippon rinsho. Japanese journal of clinical medicine, (1996 Jul) 54 (7) 1881-7. Ref: 31  
 Journal code: 0420546. ISSN: 0047-1852.  
 CY Japan  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA Japanese  
 FS Priority Journals  
 EM 199611  
 ED Entered STN: 19961219  
 Last Updated on STN: 19961219  
 Entered Medline: 19961126  
 AB Cell numbers are controlled by a homeostatic mechanism between cell growth, arrest and programmed cell death (apoptosis) in normal and cancerous tissues. One of the tumor suppressor genes, **p53**, functions as a transcription factor or transcriptional regulator through DNA and protein binding properties, and plays an important role in regulating cell cycle and induction of apoptosis. Although there are two apoptotic pathways, **p53**-independent and **p53**-dependent, the latter will be emphasized and discussed in this section. Since **p53** is often inactivated due to mutation in human cancers, understanding the **p53**-dependent apoptotic pathway is extremely important. Analysis of **p53**-dependent apoptosis as well as apoptosis caused by other **p53**-related genes should provide a clue to a new strategy for cancer therapy.

L4 ANSWER 5 OF 273 MEDLINE on STN  
 AN 96438659 MEDLINE  
 DN PubMed ID: 8841019  
 TI Structure and function of the **p53** tumor suppressor gene: clues for rational cancer therapeutic strategies.  
 AU Harris C C  
 CS Laboratory of Human Carcinogenesis, Division of Basic Science, National Cancer Institute, Bethesda, MD 20892-4255, USA.  
 SO Journal of the National Cancer Institute, (1996 Oct 16) 88 (20) 1442-55. Ref: 288  
 Journal code: 7503089. ISSN: 0027-8874.  
 CY United States  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA English  
 FS Priority Journals  
 EM 199611  
 ED Entered STN: 19961219  
 Last Updated on STN: 19961219  
 Entered Medline: 19961106  
 AB The **p53** tumor suppressor protein is involved in multiple central cellular processes, including transcription, DNA repair, genomic stability, senescence, cell cycle control, and apoptosis. **p53** is functionally inactivated by structural mutations, interaction with viral products, and endogenous cellular mechanisms in the majority of human cancers. This functional inactivation can, in some circumstances, produce resistance to DNA-damaging agents commonly used in cancer chemotherapy and radiotherapeutic approaches. Current research is defining the biochemical pathways through which **p53** induces cell cycle arrest and apoptosis. Knowledge of these fundamental processes is leading to the identification of molecular targets toward which multimodality cancer therapies, using chemotherapeutic, immunotherapeutic, and gene-therapeutic strategies, can be based.

L4 ANSWER 6 OF 273 MEDLINE on STN  
 AN 96203992 MEDLINE  
 DN PubMed ID: 8622853  
 TI New insights into **p53** function from structural studies.  
 AU Arrowsmith C H; Morin P  
 CS Division of Molecular and Structural Biology, Ontario Cancer Institute, University of Toronto, Canada.  
 SO Oncogene, (1996 Apr 4) 12 (7) 1379-85. Ref: 59  
 Journal code: 8711562. ISSN: 0950-9232.  
 CY ENGLAND: United Kingdom  
 DT Journal; Article; (JOURNAL ARTICLE)  
 General Review; (REVIEW)  
 (REVIEW, TUTORIAL)  
 LA English  
 FS Priority Journals  
 EM 199606  
 ED Entered STN: 19960627  
 Last Updated on STN: 19960627  
 Entered Medline: 19960618  
 AB Recent structural analysis of **p53** has greatly enhanced our understanding of the biochemical activities of this protein by presenting us with a detailed picture of the chemical groups in the protein that are involved in protein stability, conformation and functional interactions. The current structures form the basis for the design of potential therapeutics which could, for example, revert a DNA-binding mutant back to a DNA-binding competent conformation. The structure of the tet domain forms the basis for designing an active therapeutic **p53** with an oligomerization domain which would not cross react with a DNA-binding mutant **p53**. However, as useful as these structures have been in providing insight into the structure/function relationship for **p53**, a complete understanding of this protein awaits more detailed information on the full-length protein. In this respect, one of the most useful roles for future structural studies will be to help identify the nature of the conformational transition between latent and active

p53, and how it can be modulated.

L4 ANSWER 7 OF 273 MEDLINE on STN  
AN 96288984 MEDLINE  
DN PubMed ID: 8710022  
TI [Hereditary mutations in the p53 tumor suppressor gene; significance for clinical practice. National Work Group Hereditary Mamma Carcinoma].  
Erfelijke mutaties in het p53-tumorsuppressorgen; betekenis voor de klinische praktijk. Landelijke Werkgroep Erfelijk Mammacarcinoom.  
AU Menko F H; Nooy M A; Vasen H F  
CS Academisch Ziekenhuis Vrije Universiteit, afd. Klinische Genetica, Amsterdam.  
SO Nederlands tijdschrift voor geneeskunde, (1996 Jun 29) 140 (26) 1347-50. Ref: 21  
Journal code: 0400770. ISSN: 0028-2162.  
CY Netherlands  
DT Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
(REVIEW, TUTORIAL)  
LA Dutch  
FS Priority Journals  
EM 199609  
ED Entered STN: 19960919  
Last Updated on STN: 19960919  
Entered Medline: 19960912

L4 ANSWER 8 OF 273 MEDLINE on STN  
AN 97069847 MEDLINE  
DN PubMed ID: 8912827  
TI Lymphoepithelial carcinoma of the larynx and hypopharynx: study of eight cases with relationship to Epstein-Barr virus and p53 gene alterations, and review of the literature.  
AU MacMillan C; Kapadia S B; Finkelstein S D; Nalesnik M A; Barnes L  
CS Department of Pathology, University of Pittsburgh Medical Center, PA, USA.  
SO Human pathology, (1996 Nov) 27 (11) 1172-9. Ref: 44  
Journal code: 9421547. ISSN: 0046-8177.  
CY United States  
DT (CASE REPORTS)  
Journal; Article; (JOURNAL ARTICLE)  
General Review; (REVIEW)  
(REVIEW OF REPORTED CASES)  
LA English  
FS Priority Journals  
EM 199701  
ED Entered STN: 19970128  
Last Updated on STN: 19970128  
Entered Medline: 19970107

AB Eight cases of lymphoepithelial carcinoma (LEC) of the larynx and hypopharynx were evaluated for clinicopathologic features, and the presence of the Epstein-Barr virus (EBV) and p53 alterations. The seven men and one woman, all of non-Asian descent, averaged 64 years of age. Eighty-eight percent had histologically confirmed cervical lymph node metastasis at diagnosis. None had systemic disease. Seven of eight patients available for follow-up (mean, 17.7 months) were alive and free of disease, although one did develop recurrent tumor in the neck. Four tumors were composed, histologically, of pure LEC. Four others had foci of both LEC and conventional squamous cell carcinoma. All eight tumors exhibited alterations in p53 expression, but none was positive for EBV. Combining these 8 cases with the 15 previously published cases in the English literature indicate that LEC in this site is a rare, rather aggressive tumor, primarily of older adults (mean, 62 years) with a propensity for early cervical lymph node metastasis and eventual distant dissemination and death from disease in about one third of patients. Although p53 alterations are common and of no apparent prognostic significance, LEC at this site seems to have little, if any, relationship to the EBV in patients of non-Asian origin.

L4 ANSWER 9 OF 273 MEDLINE on STN

AN 96206040 MEDLINE  
DN PubMed ID: 8654922  
TI **p53**: puzzle and paradigm.  
AU Ko L J; Prives C  
CS Department of Biological Sciences, Columbia University, New York, New York  
10027, USA.  
NC CA58316 (NCI)  
SO Genes & development, (1996 May 1) 10 (9) 1054-72. Ref: 245  
Journal code: 8711660. ISSN: 0890-9369.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
**General Review; (REVIEW)**  
(REVIEW, ACADEMIC)  
LA English  
FS Priority Journals  
EM 199607  
ED Entered STN: 19960808  
Last Updated on STN: 19970203  
Entered Medline: 19960726

Q11 426.61166

L4 ANSWER 10 OF 273 MEDLINE on STN  
AN 96247678 MEDLINE  
DN PubMed ID: 8644842  
TI The two faces of tumor suppressor **p53**.  
AU Smith M L; Fornace A J Jr  
CS Laboratory of Molecular Pharmacology, Developmental Therapeutics Program,  
National Cancer Institute, Bethesda, Maryland 20892, USA.  
SO American journal of pathology, (1996 Apr) 148 (4) 1019-22. Ref:  
39  
Journal code: 0370502. ISSN: 0002-9440.  
CY United States  
DT Journal; Article; (JOURNAL ARTICLE)  
**General Review; (REVIEW)**  
(REVIEW, TUTORIAL)  
LA English  
FS Abridged Index Medicus Journals; Priority Journals  
EM 199607  
ED Entered STN: 19960726  
Last Updated on STN: 19960726  
Entered Medline: 19960712